

Public Health Briefs

Passive Smoking and 20-Year Cardiovascular Disease Mortality among Nonsmoking Wives, Evans County, Georgia

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Abstract: The association of passive smoking and cardiovascular disease (CVD) mortality was assessed in a cohort of 513 rural, married Black and White women who were disease-free and self-described as never-smokers at baseline in 1960. Over a 20-year period, 76 of 147 total deaths were attributed to CVD. Relative risk estimates adjusted for age, cholesterol, blood pressure, and body mass from proportional hazards models were 1.59 for CVD (95% CI = 0.99, 2.57) and 1.39 (CI = 0.99, 1.94) for all cause mortality among women with husbands who smoked cigarettes. (*Am J Public Health* 1990; 80:599-601.)

Introduction

Cardiovascular diseases account for about one-half of all deaths in the United States annually.¹ Although active smoking is well-established as a CVD risk factor,² the risk for all CVD mortality associated with passive smoking among nonsmokers has not been previously investigated. Recent studies of risks for coronary heart disease,³⁻⁸ stroke,^{4,5} or all cause mortality^{7,9,10} associated with passive smoking generally have reported weak and/or statistically nonsignificant results.

The 20-year mortality experience of nonsmoking women in Evans County, Georgia was used to assess the association of passive smoking with CVD and all cause mortality. This is the first report that includes data on both Blacks and Whites and on the consistency of self-reported smoking behaviors over time.

Methods

In 1960-61, 92 percent of all residents ages 40-74 years and a 50 percent sample of individuals ages 15-39 years in Evans County, Georgia participated in a cardiovascular disease study that included risk factor measurements, complete physical examinations, and a demographic and medical history interview.¹¹ Detailed descriptions of the Evans County study design and the 20-year mortality follow-up of the cohort have been reported elsewhere.¹¹⁻¹³ At baseline, 554 (82 percent) White and 389 (83 percent) Black women,

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among a total of the 1,127 women ages 40-74, reported that they had never smoked. The present study was restricted to the 328 White women and 185 Black older women who had never smoked and were married to male examinees who reported they either had never smoked or were current smokers at baseline. Women married to ex-smokers were excluded from the analyses as the probability for misclassification of these subjects' own smoking habits and those of their husbands was judged to be higher than for spouses of never smokers.¹⁴ A second survey of study subjects in 1967 provides data on the stability of reported smoking status.

Vital status was determined as of May 1, 1980. Underlying cause of death was abstracted from death certificates with codes 390-456 (ICD 8th Revision) defining CVD. All CVD mortality was chosen as an endpoint given the limitations of death certificate data and the small number of deaths attributed to each specific CVD entity.¹⁵ Three subjects who did not have follow-up information were excluded.

Analyses for White women were stratified by social status because of its inverse relationship with smoking status and CVD mortality in this cohort.¹⁶ White women were divided into high social status and low social status groups based on the median of the McGuire-White index of social status for all Evans County Whites. This index, based on occupation, level of education, and source of income of the head of household, was developed for use in rural settings.¹⁵ Since only 5 percent of the Black women in the Evans County population had a social status score above the median for Whites, Blacks were not stratified by social status. Exposure to passive smoking was defined by husband's smoking status (current, never) at the time of the baseline interview.

Mean baseline characteristics by passive smoke exposure were compared using t-tests. Cox proportional hazards models¹⁶ were used to estimate the association of passive smoking with time to all CVD, smoking-related CVD, and all cause mortality in this population while adjusting for age alone and for age, systolic blood pressure, serum cholesterol, body mass index (BMI), and a quadratic term for BMI. Relative risks (RR) and 95% confidence intervals (CI) were calculated using the SAS proportional hazards (PHGLM) modeling procedures,^{17,18} and the statistical significance of trends was tested using a method proposed by Rothman.¹⁹ Constancy of the relative risks over time was verified before the proportional hazards were modeled.

Results

Among nonsmoking married women, there were 179 (55 percent) of 328 White women and 117 (63 percent) of 185 Black women whose husbands reported current cigarette

TABLE 1—Mean and Standard Error of Baseline Characteristics by Passive Smoking Status of Nonsmoking Wives, Ages 40–74 Years, Evans County, Georgia, 1960–81

	White Women					
	High Social Status*		Low Social Status*		Black Women	
	Exposed	Unexposed	Exposed	Unexposed	Exposed	Unexposed
N	(78)	(83)	(101)	(66)	(117)	(68)
Age	51.9 ± 1.0	54.9 ± 0.9	52.1 ± 0.8	53.9 ± 0.9	50.3 ± 0.7	55.5 ± 1.0
Systolic Pressure	145.5 ± 3.1	150.6 ± 2.9	151.6 ± 2.9	157.6 ± 4.3	170.6 ± 3.4	176.5 ± 5.0
Diastolic Pressure	88.4 ± 1.6	90.6 ± 1.4	92.2 ± 1.3	93.1 ± 1.7	103.1 ± 1.9	103.9 ± 2.5
Serum Cholesterol	231.9 ± 4.9	237.5 ± 4.5	227.0 ± 4.4	235.7 ± 7.3	216.5 ± 3.9	216.2 ± 4.6
Body Mass Index	26.3 ± 0.6	26.4 ± 0.6	27.0 ± 0.5	28.6 ± 0.9	29.2 ± 0.6	30.0 ± 0.9

*Based on the median of McGuire-White scores for all White subjects.

smoking behavior. Among both Black and White women there were no statistically significant ($p < 0.05$) differences by passive smoking status for systolic or diastolic blood pressure, serum cholesterol or body mass (Table 1). However, passively exposed Black women and high social status White women were younger on average than nonexposed wives by 5.2 years (95% CI = 3.0, 7.6) and 3.0 years (95% CI = 0.3, 5.5), respectively. For all Whites combined, nonexposed women were also more likely to be above the median SES (socioeconomic status) level than passively exposed women (55.7 percent vs 43.6 percent).

Comparison of self-reported smoking status in 1960 and 1967 showed 98 percent of wives again reported themselves as never having smoked in 1967. Similarly, 98 percent of never smoking husbands maintained their reported status in 1967, while 25 percent of husbands who smoked in 1960 described themselves as non-smokers in 1967.

Age-adjusted RRs for all CVD, smoking-related CVD, and all cause mortality among passively exposed wives were elevated in Blacks and high social status Whites and for all subjects combined (Table 2). The opposite relationship of mortality with passive smoking status was found for low social status White women. Adjustment for other established CVD risk factors (blood pressure, cholesterol, and BMI) generally caused modest elevations of the risk estimates (Table 3) but as with the age-adjusted estimates, the confidence intervals for all subject groups included unity. A trend in risk over level of husband's smoking as reported in 1960 was only seen among high social status Whites; RRs for both total and smoking-related CVD mortality among wives whose husbands smoked <10, 10–20,

TABLE 3—Relative Risks* and 95% Confidence Intervals for Total CVD, Smoking-related† CVD, and All Cause Mortality for Wives Exposed to Passive Smoke in Evans County, Georgia, 1960–80

Cause of Death	All Subjects	Blacks	Whites	
			HSS**	LSS††
CVD Total	RR 1.59 95% CI 0.99, 2.57	1.78 0.86, 3.71	1.97 0.72, 5.34	0.79 0.32, 1.96
Smoking-related	RR 1.54 95% CI 0.93, 2.55	1.68 0.76, 3.71	1.97 0.72, 5.34	0.82 0.31, 2.15
All cause	RR 1.39 95% CI 0.99, 1.94	1.33 0.78, 2.28	1.97 1.00, 3.90	0.87 0.48, 1.59

*Hazard ratios adjusted for age, diastolic blood pressure, total serum cholesterol, body mass index (BMI = kg/meter²), and BMI².

†ICD8 codes 410–456

**High social status

††Low social status

and >20 cigarettes per day as compared to wives of nonsmokers were 1.02, 2.11, and 2.55, respectively (p for trend < 0.06). A marginally significant ($p < 0.09$) trend in risk for all CVD and smoking-related CVD over crude levels of duration of exposure was also apparent only among high social status White women.

Discussion

These data suggest an elevation of risk for death from CVD and all causes among non-smoking married women whose husbands described themselves as current smokers at the beginning of a 20-year follow-up period. Our findings for Blacks are the first report associating CVD with passive smoking in this racial group. Our observations that social status may modify the effect of passive smoke exposure may be due to chance, but a similar pattern of results for coronary heart disease (CHD) has been reported in other studies of passive smoking. Nonsignificant ($p > 0.05$) two-fold RRs for CHD among passive smokers were reported from studies of middle-class and upper-middle-class women⁶ and men⁷ while CHD risk was significantly but more modestly increased ($RR = 1.2$) among a much larger sample of predominantly blue collar Washington County, Maryland women.⁸ No increased risk for CHD was reported among public hospital patients whose husbands smoked in four British hospital regions.⁹

It is unlikely that these results can be explained by a change in smoking habits since the minimum age of these women in 1960 was 40. We lack data to examine whether exposure status changed during follow-up due to remarriage. The absence of elevated risk among exposed low social status

TABLE 2—Age-adjusted Relative Risks and 95% Confidence Intervals for Total CVD, Smoking-related* CVD, and All-Cause Mortality for Wives Exposed to Passive Smoke in Evans County, Georgia, 1960–80

Cause of Death	All Subjects	Blacks	Whites	
			HSS*	LSS**
CVD Total	RR 1.34 95% CI 0.84, 2.21	1.69 0.83, 3.46	1.66 0.64, 4.32	0.60 0.27, 1.34
Smoking-related	RR 1.29 95% CI 0.79, 2.10	1.57 0.73, 3.37	1.67 0.64, 4.36	0.61 0.25, 1.47
All cause	RR 1.31 95% CI 0.95, 1.82	1.34 0.79, 2.28	1.80 0.94, 3.47	0.72 0.41, 1.27

*ICD8 codes 410–456

**High social status

††Low social status

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White women may reflect a failure of our passive exposure index to measure exposure within the lower social stratum. Power to test for small differences in effect of passive smoking by race or social standing was lacking as were data to evaluate the role of other variables such as alcohol use or physical activity. Taken together with the results of previous studies^{8,10} and laboratory results suggesting that passive smoke exposure causes decreases in energy production in the mitochondria of heart muscle²⁰ and increased platelet aggregability in nonsmokers,²¹ our results support the health hazards of exposure to passive smoke.²⁰

ACKNOWLEDGMENTS

This work was supported by NIH grants S-T32-HL07055-13 and 2-R01-MH03341 (Merit Award). The authors thank Judson Wells for his helpful comments. The results described here were originally presented at the annual meeting of the Cardiovascular Behavioral Medicine, Epidemiology and Biostatistics Training Session in San Francisco, California, on March 29, 1989.

REFERENCES

1. Fraser GE: Preventive Cardiology. New York: Oxford University Press, 1986; 3.
2. US Department of Health and Human Services: The Health Consequences of Smoking for Women: A Report of the Surgeon General. Washington, DC: Govt Printing Office, 1980.
3. Hirayama T: Passive smoking—A new target of epidemiology. *Tokai J Exp Clin Med* 1985; 10:287-293.
4. Gillis CR, Hole DJ, Hawthorne VM, Boyle P: The effect of environmental tobacco smoke in two urban communities in the west of Scotland. *Eur J Respir Dis* 1984; 65 (suppl 133):121-126.
5. Lee PN, Chamberlain J, Alderson MR: Relationship of passive smoking to risk of lung cancer and other smoking associated diseases. *Br J Cancer* 1986; 54:97-105.
6. Garland C, Barrett-Connor E, Suarez L, Criqui MH, Wingard DL: Effects of passive smoking on ischemic heart disease mortality of nonsmokers: a prospective study. *Am J Epidemiol* 1985; 121:645-650.
7. Svendsen KH, Kuller LH, Martin MJ, Ockene JK: Effects of passive smoking in the Multiple Risk Factor Intervention Trial. *Am J Epidemiol* 1987; 126:783-795.
8. Helsing KJ, Sandler DP, Comstock GW, Chee E: Heart disease mortality in nonsmokers living with smokers. *Am J Epidemiol* 1988; 127:915-922.
9. Vandenbroucke JP, Verhersen JHH, DeBruin A, Mauritz BJ, Van Der Heide-Wessel C, Van Der Heide RM: Active and passive smoking in married couples: Results of 25 year follow-up. *Br Med J* 1984; 288:1801-1802.
10. Sandler DP, Comstock GW, Helsing KJ, Shore DL: Deaths from all causes in nonsmokers who lived with smokers. *Am J Public Health* 1989; 79:163-167.
11. Cornoni JC, Waller LE, Cassel JC, et al: The incidence study—study design and methods. *Arch Intern Med* 1971; 128:896-900.
12. Johnson JL, Heineman EF, Heiss G, Hames CG, Tyroler HA: Cardiovascular disease risk factors and mortality among Black women and White women aged 40-64 years in Evans County, Georgia. *Am J Epidemiol* 1986; 123:209-220.
13. Tyroler HA, Knowles MG, Wing SM, et al: Ischemic heart disease risk factors and twenty-year mortality in middle-age Evans County Black men. *Am Heart J* 1984; 108:738-746.
14. National Research Council: Environmental Tobacco Smoke—Measuring Exposures and Assessing Health Effects. Washington, DC: National Academy Press, 1986; 234-240.
15. McGuire C, White GD: The measurement of social status. Research paper in human development No. 3 (revised). Department of Educational Psychology, University of Texas, Austin, 1955.
16. Cox DR: Regression models and life tables. *J R Stat Soc, series B* 1972; 34:198-220.
17. Harrell FE: PHGLM procedure. Department of Clinical Biostatistics, Duke University, Durham, NC.
18. SAS Institute Inc: SAS, Release 5.18. Cary, NC: SAS Institute Inc, 1988.
19. Rothman K: Modern Epidemiology. Boston: Little, Brown and Co, 1986; 346-349.
20. Gvozdjakova A, Bada V, Sany L, et al: Smoke cardiomyopathy: disturbance of oxidative processes in myocardial mitochondria. *Cardiovas Res* 1984; 18:229-232.
21. Burghuber OC, Punzengruber CH, Sinzinger H, et al: Platelet sensitivity to prostacyclin in smokers and non-smokers. *Chest* 1986; 90:34-38.
22. Wells AJ: An estimate of adult mortality in the United States from passive smoking. *Environ Int* 1988; 14:249-265.

Community Impact of a Localized Smoking Cessation Contest

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Abstract: The present study assessed the effectiveness of a localized community contest timed to coincide with a statewide smoking cessation contest. Follow-up interviews were conducted with 218 local contest participants and 198 participants from the statewide contest. Overall cessation impact (participation rate \times abstinence) was 0.39 percent for the local contest and 0.09 percent for the statewide contest. Localized community contests offered in conjunction with statewide or national campaigns may represent cost-effective methods of reaching large numbers of smokers. (*Am J Public Health* 1990;80:601-603.)

Introduction

Contests to promote smoking cessation appear to represent cost-effective means of producing quit attempts in

community settings.¹⁻³ Quit smoking contests have been offered on a number of occasions as part of the smoking intervention in the Minnesota Heart Health Program (MHHP), a 10-year research and demonstration project intended to reduce the prevalence of heart disease.^{4,5}

Several smoking cessation contests have been timed to coincide with the Great American Smokeout, conducted annually by the American Cancer Society (referred to as "D-Day" in Minnesota). The present study examined contest participation and outcome for samples of Twin Cities area residents in the 1984 Minnesota D-Day contest. Participants from one of the intervention communities (Bloomington) were compared with a random sample of those from other Minneapolis suburbs (not within the immediate Bloomington area). It was hypothesized that the overall impact of a contest, measured by participation and abstinence outcome, offered in conjunction with specific localized community recruitment and prizes would be greater than that of the statewide contest alone.

Method

Subjects were recruited for a statewide D-Day contest during the Fall of 1984. Recruitment began August 25, 1984

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